

IRREGULARITIES OF THE HEART RESULTING FROM
DISTURBED CONDUCTIVITY.¹

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SINCE the publication, in 1883, of Gaskell's experiments upon the heart of the tortoise,² the view has prevailed in physiology that the *primum movens* of the cold-blooded heart is located in or near the mouths of the great veins. Here the property of automatic rhythmicity is most highly developed, and here, therefore, arise the impulses which set the pace of the whole heart. These impulses arise at regular intervals near the mouths of the great veins and sweep over the heart, causing sinus venosus, auricles, ventricle, and bulbus arteriosus to contract successively in the order here given.

The presentation by Gaskell of an abundance of evidence proved these fundamental facts. For our purpose it will suffice, however, to refer to but one phase of this work, namely, that dealing with the blocking of the impulse on its way through the heart. This may be accomplished, as was shown by Gaskell, in one of two ways: (1) The impulse may be blocked gradually by cutting across the heart transversely at any level. When this is done carefully and gradually a stage is eventually reached in which that part of the heart located below the cut (this may be conveniently termed the distal heart) will beat perceptibly later than the part above (the proximal heart) with which it is now connected by a relatively narrow bridge of intact heart tissue. With further narrowing of the connecting bridge a stage may be obtained in which the distal heart fails to contract with every other beat of the proximal heart. The rate of the latter is to that of the former as 2 is to 1; to apply a term that has come into general use, the rhythm is 2 to 1. Higher grades of block, 3 to 1 and 4 to 1 rhythms, may also be obtained, but, with further cutting, the block to the passage of the impulse soon becomes complete despite incomplete anatomical separation of the two parts of the heart. The impulse starting in the more rhythmical parts of the heart can no longer reach the distal heart, and the latter, therefore, ceases to beat until it can, in case it beats at all, develop its inherent rhythmicity. Its rate of beat is then always slower than that of the proximal heart, it matters not through what level of the heart the

¹ Read by invitation at a meeting of the Section on General Medicine of the College of Physicians of Philadelphia, January 13, 1908.

² Jour. Physiol., 1883, iv, 43.

cut may have been made. A similar stoppage of the distal heart with subsequent development of rhythm is seen when the cut is completed rapidly. Then, of course, none of the intermediate stages of block intervene. (2) The impulse may be blocked if instead of cutting the heart tissue, it be gradually compressed in a clamp.³ Identical results are obtained with both methods.

These experiments teach (a) that the upper parts of the cold-blooded heart are more rhythmical than the lower, (b) that the pace of the whole heart is normally set by the upper more rhythmical parts, and (c) that at the moment a less rhythmical part of the heart is severed physiologically from a more rhythmical part, the former at first stops beating and then, in case it possesses the property of automatic rhythmicity, it gradually assumes this function and begins to beat, otherwise it remains quiescent indefinitely.

Since the slightest obvious interference with the conduction of the impulse between any two parts of the heart manifests itself as an interval between the beats of such parts, Gaskell has concluded that in the normal heart the perceptible interval between the beats of the several chambers, sinus and auricles, auricles and ventricle, must be due to the existence in their junctions of a natural block to the passage of the impulse. The heart tissue in these places, he has pointed out, differs in structure and arrangement from those parts of the heart tissue that constitute the walls of the several chambers. These differences, in his opinion, suffice to account for the delay in the transmission of the impulse from chamber to chamber.

It is not a difficult matter to picture to ourselves in the case of the cold-blooded heart the irregularities of heart beat due to experimentally disturbed conductivity. Furthermore, the heart of cold-blooded animals lends itself well to experiments of the kind described above. Not only is it relatively simple structurally in that developmentally it is not far removed from the simple primitive heart tube, but in addition the heart possesses such a high grade of automatic rhythmicity that it may be removed from the organism and experimented upon as described and still continue to beat.

The conditions with which the experimenter has to deal in the case of the warm-blooded heart and particularly in the case of the mammalian heart are not quite so simple. Nevertheless, when due allowance is made for differences in structure and when appropriate methods of experimentation are employed, it can be shown that interference with the conduction of the excitation wave has much the same effects upon the heart beat in both classes of animals.

Structurally the mammalian heart differs materially from the cold-blooded heart. An anatomically distinct sinus venosus no longer exists, and there are present in it two completely separated auricles

³ Gaskell, *Philosophical Trans.*, pt. III.

and ventricles. Since, however, the mammalian heart is evolved from the cold-blooded heart it should be possible for embryologists to determine their analogous parts. The problem is an old one. Very recently, however, Keith and Flack,⁴ stimulated, apparently by the active interest taken in the subject by physiologists and experimental pathologists, have studied very carefully the comparative anatomy of the human heart and especially of those parts of the human heart which correspond with the junctions in the more primitive types. The results recorded are of the greatest interest and value to students of the manifestations of disturbed conductivity in the heart of man, and we may, therefore, be justified in quoting at some length the significant conclusions of their research.

Keith and Flack first call attention to the fact that from the standpoint of comparative anatomy the typical mammalian heart may be divided into (1) the sinus venosus, (2) the auricular canal, (3) the auricle, (4) the ventricle, and (5) the bulbus cordis. "In the human and mammalian heart the musculature of the auricular canal has grown over and submerged the greater part of the sinus; two parts only are left exposed on the surface of the heart: (1) The musculature of the superior vena cava, and (2) the musculature of the coronary sinus." But on section "a second or deep stratum of the musculature is seen; this probably belongs to the sinus venosus, since it extends beneath the endocardium of the auricle, from the position of one venous valve to that of the other." There is also a part of the musculature "in the Thebesian and Eustachian valves, these being remnants of the right venous valves. There is often also to be found a thin muscular layer along the lower border of the fossa ovalis. . . ." All of these remnants of the sinus are in the right auricle. "It may be, however, that there are also remnants of the sinus in the left auricle of the human heart" around the orifices of the pulmonary veins. "Thus in the right auricle of the human heart there is musculature derived from three sources: (1) From the auricle proper, (2) from the auricular canal, (3) from the sinus venosus. . . . All of these parts are in the freest muscular continuity."

"In the mammalian heart a distinct remnant of the sino-auricular junction, so well shown in more primitive hearts, can be recognized." Thus "in the human heart . . . an artery or arterial circle lies in the junction; the artery is surrounded by fibrous tissue in which are numerous peculiar muscle fibers, some nerve cells, and nerve fibers." "The muscle here has a structure quite similar to that of the auriculoventricular bundle."

The question whether there exists an anatomically distinct junction between the musculatures of the auricles is not considered by Keith and Flack. But all anatomists, so far as I am aware,

⁴ Jour. Anat. and Physiol., 1907, xli, 172.

describe direct continuity of a large part of the muscle fibers of the two auricles.

It is hardly necessary to refer here, owing to the interest Philadelphians have taken in this subject from the very beginning, to the recent researches on the anatomy of the auriculoventricular junction. That the auriculoventricular bundle constitutes the only bit of heart tissue joining the heart tissue of the auricles with that of the ventricles is a fact that is now universally accepted. Keith and Flack describe the auriculoventricular bundle as a system of fibers which "begins in the 'knoten,' a small mass of interwoven fibers in the central tendon of the heart, having slender connections with (1) the musculature of the interauricular septum, and (2) the circular fibers of the right auricular canal. From this arises the main bundle which passes along the upper border of the inter-ventricular septum below the *pars membranacea septi*. Here it divides into a right and a left division which pursue a subendocardial course in the right and the left ventricles respectively, and, finally, fuse with the ventricular muscle."

There is no line of demarcation between the musculatures of the two ventricles. The work of MacCallum² has demonstrated conclusively that the ventricles, "instead of being a complex of fibers running at random, is merely a simple, long band of muscle with a tendon at each end as in other muscles." This band has its origin in the connective tissue of the auriculoventricular junction upon one side of the heart, passes around the outer wall of that side, thence to and through the ventricular septum, encircles the outer wall of the other ventricle and terminates in the *chordæ tendinæ* of that side. The musculature of the ventricles has therefore the arrangement of a scroll, and we may assume, in the absence of definite investigations upon this point, that the fibers of the auriculoventricular bundle make connection with the centre of the scroll.

Functionally the main difference between warm and cold-blooded hearts, from the standpoint of the experimenter, consists in the dependence of the former upon the coronary circulation. For physiological purposes the mammalian heart must be studied either while retaining its natural relation to the organism or when, after excision, its beat is maintained by the perfusion of its coronary system.

The studies of the anatomy of the mammalian heart outlined above should suggest to the investigator acquainted with the physiology of the cold-blooded heart that in the heart of mammals there are in all probability two natural blocking points: one at the junction of the rudiments of the sinus with the auricles (mainly in the right auricle), the other at the junction of the auricles with the ventricles, provided it can be shown that the impulse that causes the

² Johns Hopkins Hosp. Rep., 1900, ix, 307.

heart to beat starts in the sinus and traverses the auriculoventricular bundle; and it might be justifiable to infer further upon the same premise, that the natural block at the auriculoventricular junction is the more efficient. Practically all of these inferences have been justified by the test of experiment and it is with the results of such experiments that this paper principally deals.

The influence upon the heart beat of the block, both natural and experimental, has been studied most thoroughly in connection with the auriculoventricular junction, and we may therefore begin the physiological section of this paper with the presentation of the clearest subject. At the outset it may not, perhaps, be superfluous to state that the auriculoventricular bundle constitutes the only functional connection between the auricles and the ventricles of the mammalian heart. When this bundle, or practically nothing but it, is transected in the excised artificially perfused heart⁶ or when it is crushed in the heart retaining its normal relations to the organism,⁷ the ventricles usually come to rest at once, whereas the auricles continue to beat with undisturbed rate and rhythm. Soon, however, the ventricles begin to beat, slowly at first, but the rate of beat gradually increases to a constant one which is, even when fully developed, always slower than that of the auricles. Now the auricles and ventricles beat quite independently of each other; the latter more slowly than the former. The justifiable inferences from these experiments are: (1) That the pace-maker of the mammalian heart is not in the ventricles (after section of the heart tissue the proximal heart beats more rapidly than the distal)—it lies either in or above the auricles; (2) that the ventricles of the mammalian heart are automatically rhythmic, but that while they are driven by the auricles their rhythmicity is held in abeyance—it develops gradually when they are thrown upon their own resources. Furthermore, the pause between the contractions of auricles and ventricles of the normal heart (As-Vs interval) is satisfactorily accounted for by the fact that the impulse, in passing from one to the other of these regions, must traverse a long narrow bridge.

On account of the minuteness of the auriculoventricular bundle its conductivity cannot well be gradually impaired by slowly cutting into it. It may, however, be so impaired by gradually compressing it in a specially devised clamp applied to the normally beating heart.⁸ In this way all grades of impaired conductivity, such as we have described in connection with the cold-blooded heart, may be obtained. These, in order of their appearance, consist of (1) an increase in the

⁶ His, Jr. *Zntrbl. f. Physiol.*, 1895, ix, 469. Humblet: *Arch. intern. de physiol.*, 1904, i, 278. Hering (read March 10, 1905), *Prager med. Wochenschrift*, xxx, No. 14; also, *Pflüg. Arch.* 1905, lvii, 97.

⁷ Erlanger (read March 20, 1905), *Bull. Johns Hopkins Hosp.*, 1905, xvi, 202; also, *Jour. Exp. Med.*, viii, 8. v. Tabora: *Ztschr. f. exp. Path. und Therap.*, 1906, iii, 499.

⁸ Erlanger. *loc. cit.*

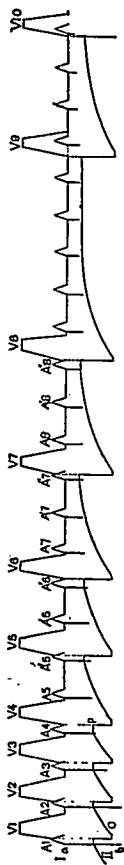


FIG. 1.—Diagram to illustrate the genesis of heart block. For description see text.

As-Vs interval. (2) The occasional failure of the ventricles to respond to the regularly contracting auricles—these “ventricular silences” may recur with great regularity, for example, after every tenth, ninth, or eighth, etc., auricular contraction. (3) The failure of the ventricles to respond to every other auricular contraction—a 2 to 1 auriculoventricular rhythm. (4) A 3 to 1 rhythm. (5) Relatively complete auriculoventricular heart block (of which more in a moment). (6) Absolutely complete auriculoventricular heart block. These several disturbances in conductivity I have attempted to make clear by means of a diagram⁹ (Fig. 1) based in part upon experimentally demonstrated facts, in part upon logical inferences from such facts. In the figure the line I shows diagrammatically the movements of the heart, A1, A2, A3, etc., being the contractions of the auricles; V1, V2, V3, etc., the contractions of the ventricles. Line II represents diagrammatically the variations in the irritability of the ventricles. It is an old observation, one dating from 1876,¹⁰ that with each contraction of heart tissue its irritability is completely annulled. During the phase of contraction heart tissue does not respond to stimulation, but during the phase of relaxation and during the pause following it the irritability returns and then gradually increases. The increase, rather rapid at first, becomes slower and slower as the irritability approaches its maximum level. The curve of irritability included between successive ventricular contractions tends, therefore, to approach the level of maximum irritability asymptotically, that is to say, it tends to become parallel to the base line. These variations in the irritability of heart muscle are explained as follows: The resting heart tissue stores within itself an unstable energy yielding material, the instability of which increases as it accumulates. A stimulus strong enough to elicit a contraction does so by decomposing all of this material. The irritability of the heart tissue, it may be assumed, depends

⁹ Erlanger. *Amer. Jour. Physiol.*, 1906, xvi, 160.

¹⁰ Marey. *Trav. d. Lab.*, 1876, 73.

upon the amount of the unstable substance present in it at any one time. Immediately after the heart has contracted it, therefore, contains none of the unstable substance; consequently it is non-irritable. But after the heart has completed its contraction the material begins to accumulate rapidly at first and then more and more slowly; the irritability, when it returns, therefore increases rapidly at first but finally becomes more or less constant. Or stated in another way, shortly after the completion of a contraction, a stimulus to be efficient must be relatively strong, but as time passes a relatively weaker stimulus becomes effective. Indeed what has been termed the inner stimulus may, in case the heart tissue is not otherwise disturbed, become efficient and cause the heart to beat spontaneously. It is justifiable to assume further, that the strength of the impulse starting in any part of the heart depends upon the amount of the unstable material decomposed by the contraction associated with it. In other words, the strength of the impulse in general varies with the interval between contractions. Finally the latent period of contraction of heart tissue varies inversely as the strength of the stimulus producing the contraction, and inversely as the irritability of the heart tissue. That is to say, a relatively strong stimulus produces a contraction with a short latent period, while in the case of a stimulus of a given strength acting upon a more or less irritable tissue, the resulting contraction will in the former case have a short latent period, in the latter a long latent period.

I do not wish to give the impression that the factors taken into consideration in the development of this theory are the only ones that play a part in determining the behavior of the heart toward interference with the transmission of the contraction wave through it. Although, apparently complex, it is the simplest explanation of the complex manifestations of heart block and it is hoped that it may prove as useful to the clinician as it has to me as a working hypothesis in the elucidation of the phenomena of heart block.

The diagram depicts the results of gradually clamping the auriculo-ventricular bundle. Pressure exerted upon the bundle will have the effect of reducing the efficiency of the auricular impulse that reaches the ventricles. The efficiency of this impulse is represented by the length of the heavy line dropped from the foot of the auricular wave. It is assumed that this impulse is efficient whenever the line representing it intersects the curve of irritability of the ventricles (II). A ventricular contraction then results. The duration of the latent period of the ventricular contraction is represented by the length of the horizontal line at the crest of the waves of irritability (on II).

We are now in a position to explain with the aid of the diagram the various grades and phases of disturbed conductivity at the

auriculoventricular junction. The first two cardiac cycles¹ are normal. The impulse (*ab*) descending from the auricles (A1 and A2) into the ventricles are much stronger than a barely efficient one. The latent period of ventricular contraction is short.

The rest of the diagram depicts some of the effects of gradually clamping the auriculoventricular bundle. The first effect is to diminish the strength of the impulse (line descending from foot of A3); it is still, however, efficient (intersects the curve of irritability of the ventricles II), but, the stimulus being weaker, the latent period of contraction of the ventricles—the As-Vs interval—is longer. The same is true in the case of the fourth cardiac cycle (A4, V4) the only difference being a still greater divergence from the normal. The auricular impulse (A5) of the fifth cardiac cycle (A5, A'5, V5), owing to the increase in the resistance to its passage through the auriculoventricular bundle, does not reach the curve of irritability of the ventricles; in other words this impulse is sub-minimal. The ventricles, therefore, do not contract and their irritability instead of falling to 0 continues to increase. On account of this increased irritability the impulse associated with the next auricular beat (A'5), although no stronger than that of A5, is efficient and the ventricles contract. The latent period of the ventricular contraction is shorter than that of the preceding one (V4) for the reason that the irritability of the ventricles is higher while the efficiency of the auricular impulse (A'5) is still relatively great. A continuance of this state of affairs would result in a lasting 2 to 1 auriculoventricular rhythm. A slightly greater block to the passage of the impulse through the auriculoventricular bundle may so alter the relation of efficiency of impulse to irritability of ventricle that the rhythm becomes 3 to 1. This is shown in cycles 7 and 8 in the diagram. Rarely, however, are higher rhythms, 4 to 1 and 5 to 1, seen in the mammalian heart. The reason for this, illustrated in the diagram, is to be found in the fact that although the auricular impulses may succeed in penetrating into the ventricles they always remain subminimal with respect to the irritability of the ventricles. They therefore cease beating until their inherent stimulus suffices to originate a contraction, when they beat automatically and quite independently of the auricles. We now have before us the condition that was termed above relatively complete block. It is relatively complete because the degree of block depends upon the efficiency of auricular impulses in relation to the irritability of the ventricles. If we could by some means increase the irritability of the ventricles the block might become partial again; so, too, might the block become partial if we could increase the efficiency of the auricular

¹ It is convenient for our purposes to define cardiac cycle as those events that intervene between the beginning of an auricular contraction immediately following a ventricular contraction, and the beginning of the auricular contraction immediately following the next ventricular contraction.

impulses. As a matter of fact it does lie within our power to vary the strength of the auricular impulse. This, we have assumed above, may be accomplished by altering the rate of the auricular beats, an increase in rate being associated with a diminution, and a decrease in rate with an increase in the efficiency of the impulse. In the latter case, for example, the impulse of the third or fourth auricular contractions following V8 might have become efficient stimuli. Then the block would have become partial, and in the former case the efficiency of the auricular impulse A''8 might have fallen below that required for stimulation of the ventricles, and the partial block then existing might have become relatively complete. The arrhythmias associated with other forms of heart block are easily comprehended when viewed in the light of this hypothesis.

SINO-AURICULAR HEART BLOCK. So far as I am aware a condition that might unequivocally be termed sino-auricular heart block has never been experimentally produced. Nevertheless the anatomical description of the heart given above would seem to indicate the possibility of there being another natural blocking point at the sino-auricular junction. Although there is in this place no narrow muscular bridge such as is found at the auriculoventricular junction still we find here interpolated between the sinus and the auricle a peculiar heart tissue. We find very little more than this at the sino-auricular junction of the cold-blooded heart, and yet most physiologists, following the lead of Gaskell, believe that it suffices to account for the S;s-As interval in that heart; it, in other words, acts in that heart as the natural block to the passage of the impulse. And it seems to me possible that under pathological conditions, such for example as occlusion of the artery lying in the sino-auricular junction of the mammalian heart, the tissue in this junction might be so impaired functionally as partially or completely to block the passage of the impulse.

However that may be, the question presents itself for consideration: Is there any evidence that in the mammalian heart the impulse arising in the *primum movens* (assuming this be in the mouths of the great veins) is materially delayed in the sino-auricular junction? In the present state of our knowledge this question cannot be answered. None of the recent workers in this field has, so far as I am aware, succeeded in satisfactorily recording the contractions of the superior vena cava simultaneously with those of the auricles. And since, according to Keith and Flack, the superior vena cava is the only part of the sinus of the mammalian heart that is not incorporated within the tissue of the auricle, the problem of determining by direct methods the duration of the normal S;s-As interval, if such interval there be, seems to be impossible of direct solution.

Another question then suggests itself, namely: Is it possible artificially to block the passage of the impulse through the region of the mammalian sinus, using for this purpose methods similar to those

employed by Gaskell in his study of conduction in the cold-blooded heart? That this can be accomplished has been demonstrated by experiments which, although indirect, are I believe none the less conclusive.¹² A block has been established in the following way: The heart of a rabbit is excised and perfused with Locke's solution. A heavy thread is passed through the right auricle entering by way of the inferior vena cava and leaving by way of the superior. After the heart has begun to beat normally gentle traction is exerted upon the projecting ends of the thread so as to elevate a cone of auricular tissue. Naturally this cone includes the mouths of the great veins and a variable amount of the tissue of the right auricle bordering upon them. While holding the thread taut the two projecting ends are twisted one upon the other. The torsion is, of course, not limited to the thread, but is continued to the auricular tissue drawn up by it. In this way, therefore, a large part if not all of the sinus region of the auricles is compressed, the greatest tension being exerted, however, upon the base of the cone. It is impossible in an experiment of this kind to record or even to see the contractions of the part of the heart twisted. Nevertheless by these means an irregularity of heart beat has been repeatedly obtained which, when viewed in the light of the remarks of the preceding section, could be caused only by the interposition of a block in the way of the impulse from a more rhythmical to a less rhythmical part of the heart. When the torsion is quickly exerted upon the sinus region of the right auricle the whole heart may apparently cease to beat just as the ventricles may cease beating when an auriculoventricular block is suddenly established. After a variable pause (in Fig. 2 the first pause lasts about six seconds) the heart may contract with normal sequence, that is, auricles first, followed in a moment by ventricles. Then there succeeds a shorter pause followed by another apparently normal contraction and so on until a constant but slow rate obtains. This period of the experiment corresponds with the development of rhythm in the ventricles after the establishment of complete auriculoventricular block, and without doubt some part of the heart without the twisted area is here developing the rhythm that determines the beat of the auricles and ventricles in lieu of the *primum movens* which the operation has rendered functionless with respect to the rest of the heart.

The constant but slow auriculoventricular rate that next obtains is in all probability the expression of complete sino-auricular block. Now, in case the treatment to which the auricle has been subjected has not permanently damaged the tissue concerned with the origination of the normal beat, the heart will, when the tension is removed, slowly but completely resume its normal state. At first, during such recovery, the visibly beating portions of the heart may

¹² Erlanger and Blackman. Amer. Jour. Physiol., 1907, xix, 125.

contract at intervals almost exactly equal to thrice the normal period, evidently 3 to 1 sino-auricular block, and then there may be seen in successive stages sino-auricular block of alternating 3 to 1 and 2 to 1, 2 to 1, alternating 2 to 1 and 1 to 1, and, finally, the normal 1 to 1 rhythms (Figs. 3, 4, and 5). That is to say, the heart periods will in the first case be alternately equal to three and two normal periods, then to two normal periods, then alternately to one and two normal periods, while, finally, the heart may beat just as it did before its rhythm had been disturbed. These irregularities, even in the absence of any direct evidence that some part of the heart above the assumed block is throughout the experiment beating at the normal rate, practically prove, in the light of the hypothesis developed above, that such is actually the case. The experiment then suggests that the *primum movens* is situated above the lowermost edge of the twisted part of the auricle. That such is the case is indicated by the fact that in the dying heart, this is the part of the heart which may show the last contractions. It is likewise the part that may first begin to beat when the quiescent heart is perfused.¹³ And in at least one of the several experiments the cone of the auricles picked up by the thread, after having been separated functionally from the rest of the heart, by means of a crush through its base, was seen to beat more rapidly than, and independently of, the other parts of the heart.¹⁴

Sino-auricular heart block, or at least partial sino-auricular heart block, is characterized then by the occurrence of heart cycles whose durations are simple multiples of that of the normal heart cycle. Admitting this to be a correct criterion, then partial sino-auricular heart block has been casually observed in man by Wenckebach¹⁵ and in the dog by Hering.¹⁶ In neither case could the exact cause of the arrhythmia be determined.

AURICULO-AURICULAR HEART BLOCK. The term auriculo-auricular heart block is self-explanatory. It is a condition in which the conductivity of the tissue joining the two auricles is depressed to such an extent that the impulse that causes the heart to beat is blocked either partially or completely on its way from one to the other auricle. Experimentally the impulse may be so blocked in the excised heart of the rabbit either by a line of crush or by means of actual separation with scissors.¹⁷

I shall consider first the effects of the functional separation of the right auricle from the left auricle plus auricular septum by means of a cut parallel to, and just to the right of, the line of junction of the auricular septum with the vault of the auricles. Let this cut be so planned that there is left between the two auricles a bridge of uncut

¹³ MacWilliam: Jour. of Physiol., 1888, ix, 167. Hering: Pflüger's Arch., 1900, lxxxi, 21.
 Fredericq: Arch. intern. d. physiol., 1906, iv, 57. Erlanger and Blackman: Loc. cit.

¹⁴ Erlanger and Blackman. Loc. cit.

¹⁵ Arch. f. Physiol., 1906, 297

¹⁶ Ztschr. f. exp Path. u. Therap., 1906, 511.

¹⁷ Erlanger and Blackman. Loc. cit.

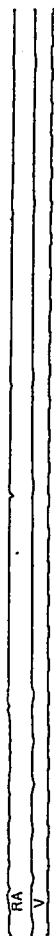


FIG. 2.—To show the production of, and the different stages of recovery from, complete sino-auricular block resulting from the twisting of the region of the great veins. Before, there was a 2 to 1 auriculoventricular block. The duration of torsion is indicated by the line just below the time record. Time-interval between two symmetrical marks is one second. Down strokes are contractions. Rabbit.

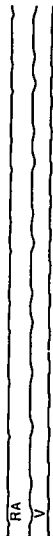


FIG. 3.—Shows the change from a sino-auricular block of the 2 to 1 type to a block of the alternate 2 to 1, 1 to 1 type. Now, none of the auricular impulses are blocked at the auriculoventricular junction.



FIG. 4.—Shows alternate 2 to 1 and 1 to 1 sino-auricular block.

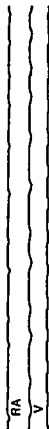


FIG. 5.—Shows the return to normal sequence from an alternate 2 to 1 and 1 to 1 sino-auricular block.

auricular tissue which, however, is damaged to such an extent that impulses cannot at first make their way across it from one auricle to the other. At the moment this is accomplished the left auricle and auricular septum may at once come to rest, whereas the right auricle continues to beat with undisturbed rate. After a short pause, however, the left auricle with attached septum begins to beat, perhaps slowly at first, but more rapidly later when its rhythmicity is fully developed. Then the left side contracts regularly, but more slowly than, and independently of, the right (Fig. 6). We have here, to coin a word, complete dextrauriculo-septolevaauricular heart block. Subsequently, with the gradual disappearance of the block, there occur in turn the several stages of partial block such as 3 to 1 dextrauriculo-septolevaauricular heart block when the left auricle plus septum contracts after every third beat of the right auricle, 2 to 1 block (Fig. 7), and then perhaps the normal rhythm with a perceptibly long interval between the contractions of the right and of the left auricle, or as we might call it by analogy, a long dAs-SelAs interval, and finally the heart beat becomes absolutely normal.¹⁸

The auriculoventricular bundle, the physiological connection between the auricles and ventricles, lies, as we have seen, in the septum. The beat of the ventricles in an experiment of the kind just described is determined, therefore, by the beats of the auricular septum, and since the connection of the left auricle with the septum is undisturbed, the left auricle and the ventricles beat with the sequence that obtains in the intact heart.

The experiments upon sino-auricular heart block described above proved the most rhythmical part of the heart to be in the right auricle in the region of the mouths of the cavæ. The experiment just described confirms this finding in that it shows the right auricle to possess a higher degree of rhythmicity than the left auricle plus the auricular septum. In addition the experiment demonstrates that the left auricle plus the septum like the right auricle and the ventricle is automatically rhythmical; for in the case of complete separation of the two parts both beat, although independently of each other—the left side always more slowly than the right.

Let us consider next the effect of separating functionally the left auricle from the right auricle plus the auricular septum, altering the method described above only in so far as the position of the cut is concerned. In this case the cut is carried through the vault of the auricles just to the left of, instead of just to the right of, the septum. The left auricle usually stops beating at the moment the functional separation becomes complete, whereas, the right auricle and septum continue to beat without the slightest change in rate (Fig. 6). The left auricle in a vast majority of experiments does not beat again as long as the functional separation is complete. In this respect the

¹⁸ Wenckebach (loc. cit.) has obtained from man tracings which he believes show the right auricle to be beating independently of the rest of the heart.



FIG. 6.—Shows at the beginning complete blocks between the right auricle and the left auricle plus the septum, and between the latter and the ventricles. Later (at irregularities), the left auricle was separated from the auricular septum and stopped beating at once. Down strokes are contractions. Rabbit.

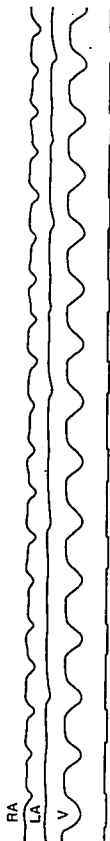


FIG. 7—Shows the ventricles beating independently and the left auricle responding at first to every fourth contraction of the right auricle, then to the third, and then to every second to the end of the tracing. Down-strokes are contractions. Rabbit.

separation on the one hand of the left auricle and upon the other of the left auricle plus the septum from the right yields different results. The difference is due to the fact that, whereas, the left auricle plus the septum possesses automatic rhythmicity, the left auricle alone does not or possesses it to but a slight degree. Evidently the septum alone is automatically rhythmical since it is the part of the heart that determines the beat of the left auricle when together they are separated from the right. That the septum is automatically rhythmical may be proved by the direct experiment of separating it from both of the auricles by cuts through the vault to either side of it. Then the septum may be seen to beat independently of the two auricles and to determine the beat of the ventricles, while the right auricle beats more rapidly than it, the left auricle remaining quiescent.

To complete the consideration of the effects of separating functionally the right auricle plus the septum from the left auricle, as the conductivity of the connecting bridge gradually returns and the impulse of the right auricle succeeds in penetrating into the left, the various stages of partial block make their appearance. And we may, therefore, see 3 to 1, 2 to 1, etc, septodextauriculo-levauricular heart block.

Many other forms of heart block have been produced in the laboratory, but those which have been described here in barest outline are the ones which, for anatomical reasons, ought to be of the most frequent occurrence in the experience of the clinician. Just as partial and complete block may be established experimentally between any two parts of the heart, provided one of the parts be spontaneously rhythmical, by narrowing the functional connection between them, so, too, the same forms of block might well be produced by pathological processes of a kind that severs the functional connection of one part with another. When a transitory block is thus established by a lesion to repeat, the less rhythmical part at first stops beating but, in case it possess spontaneous rhythmicity, it soon begins to beat again, first in complete block and then in partial block with all of the rhythms that have been observed for example in the transitory auriculoventricular block. Naturally, auriculoventricular block of all the forms will be found to be, and has been found to be, the most common clinically, not alone for the reason that the narrowness of the connection between the auricles and the ventricles makes easier its partial and complete destruction by disease, but also because the clinical manifestation of auriculoventricular heart block (Stokes-Adams disease) are unmistakable. While it may be possible in particularly clear cases of auriculoventricular heart block to make a correct diagnosis without the aid of the more exact methods of investigating the organs of the circulation, the more obscure cases of this form of heart block, but especially the other forms of heart block, can be recognized only with the aid of the graphic method.